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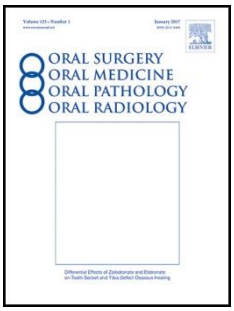

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Clinical features and presentation of oral potentially malignant disorders

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Clinical features and presentation of oral potentially premalignant disorders

Statement of Clinical Relevance

Potentially malignant Disorders (eg. Leukoplakia and erythroplakia) encountered during a routine oral mucosal examination (apart from a malignancy) represent the most significant clinical findings in a dental practice. Early diagnosis, referral to a specialist and appropriate intervention may reduce the rate of progression of these conditions to invasive cancer.

Abstract

Oral potentially malignant disorders (OPMDs) are conditions that precede the appearance of invasive cancers of the oral cavity. The term embraces both precancerous lesions and conditions referred to in the earlier WHO definitions. Leukoplakia is the most common OPMD; erythroplakia though rare is more serious. Several variants of leukoplakia are recognized and clinical subtyping could help in predicting the prognosis to a limited extent. Biopsy is essential to confirm the provisional clinical diagnosis and timely referral to a specialist is indicated. Certain OPMDs such as oral submucous fibrosis are encountered particularly in population groups from Asia with specific life-style habits. This review provides clinical descriptions of the wide range of PMDs encountered in the oral cavity as a prelude to the topics discussed in this focus issue.

Introduction

A range of oral mucosal disorders with an increased risk of malignancy is described in the literature and are listed under the umbrella of Oral Potentially Malignant Disorders, herein abbreviated to OPMD. The spectrum of OPMDs include: oral leukoplakia, erythroplakia , erythroleukoplakia, oral submucous fibrosis (OSF), palatal lesions in reverse smokers, oral lichen planus, oral lichenoid reactions, graft vs host disease, oral lupus erythematosus and some hereditary conditions such as dyskeratosis congenita and epidemolysis bullosa. Actinic cheilitis of the lower lip is also associated with an increased risk of lip cancer. The majority of these disorders may be asymptomatic in the early stages of their evolution and may be detected by dental practitioners on routine oral examination. It is essential therefore that health professionals are knowledgeable of the clinical features and diagnostic aspects of

OPMDs in order to further investigate and where appropriate make a referral to a specialist for treatment.

It has been known for over a century that oral cancer may develop in areas of preexisting mucosal pathology found in the oral cavity. In the literature these lesions were referred to by terms such as “pre-cancer”, “precancerous/ premalignant lesions”, “intra epithelial neoplasia”. A more precise term - “potentially malignant disorders” - was adopted by the WHO Collaborating Centre as there is no certainty that all precancerous lesions will eventually develop into oral cancer.[1] The term also embraces both precancerous lesions and conditions that were included in the previous WHO classification.[2] In this focus issue it is proposed to introduce a new terminology “Potentially Premalignant Oral Epithelial Lesions - PPOEL” (see accompanying editorial). The underlying concept is that these lesions, have a potential to become malignant, so, in their current state i.e. before malignant transformation, are still (potentially) premalignant.

Since the publication characterising oral potentially malignant disorders in 2007 in the past decade new evidence has emerged that supports the inclusion of oral lichenoid lesions and oral lesions of graft vs host disease as potentially malignant disorders. A brief description of these conditions is also included here.

Leukoplakia

In order to precisely diagnose this condition it is important to consider the definition given to oral leukoplakia.[3] Historically the term leukoplakia was a clinical term used to denote any adherent white patch or a plaque (keratosis). Over several decades clinicians realised that all white patches arising in the oral cavity should not be labelled as oral leukoplakia. Several definitions have been given to oral leukoplakia over the past few decades. The most recent definition in use refers to leukoplakia as ‘predominantly white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer’.[1] Examples of other benign white lesions that should be excluded to arrive at the diagnosis of oral leukoplakia are: frictional keratosis (cheek biting), alveolar ridge keratosis, leukoedema, white sponge nevus and Fordyce granules which are usually buff coloured.

- Oral leukoplakia may be asymptomatic or display a benign clinical appearance making it difficult for the clinician to sometimes differentiate them from common reactive or inflammatory (benign) disorders of the oral mucosa.
- Leukoplakias are usually diagnosed after the fourth decade of life. They are more common in males [4] and are six times more common among smokers than among non-smokers. Alcohol consumption is an independent risk factor. Leukoplakia is not associated with any chemical or physical causative agents except tobacco, alcohol or betel quid.[5] In a minority of leukoplakias human papillomavirus (HPV) may have a potential role. Some leukoplakias are idiopathic and may not have a known risk factor. The risk factors for oral leukoplakia are considered in detail in a later paper in this focus issue.
- Common sites of involvement in western industrialised populations include the lateral margin of the tongue and the floor of mouth. However, among Asian people buccal mucosa and lower buccal grooves are commonly affected due to placement of betel quid at these locations. Gingival leukoplakia (affecting gums) is uncommon but has been reported to affect predominantly the Japanese population.[6]
- A patch of oral leukoplakia may vary from a quite small and circumscribed area to an extensive lesion involving a large area of mucosa
- There are two main clinical types of leukoplakia encountered in clinical practice; homogeneous and non homogeneous leukoplakia. The distinction is based on surface colour and morphological (thickness and texture) characteristics.
    - Homogeneous leukoplakias are uniformly flat and thin, have a smooth surface and may exhibit shallow cracks.
    - Non-homogeneous varieties comprise of 3 clinical types and are usually symptomatic:
        - (1) Speckled: mixed, white and red (also termed erythroleukoplakia) but retaining predominantly white character.
        - (2) Nodular: small polypoid outgrowths, rounded red or white excrescences.
        - (3) Verrucous or exophytic: wrinkled or corrugated surface appearance.

- Generally most leukoplakias are asymptomatic and are found during a routine visual examination by a practitioner. Symptoms if present, are associated with non-homogeneous speckled variety, and in this author's experience include discomfort, tingling and sensitivity to touch, hot beverages or spicy foods. A red component in the leukoplakia (erythroleukoplakia) indicates possible colonization by candida organisms and an increased risk for dysplasia and/or malignancy.[7]
- When widespread or multiple patches of leukoplakia are noted the term proliferative verrucous leukoplakia (PVL) is used. Other criteria for diagnosis of PVL are given in a later section. This is a distinct entity that carries a higher risk for malignant transformation and is fortunately rare.
- A provisional clinical diagnosis of leukoplakia is made for a white patch after excluding a local traumatic cause, by confirming that it cannot be scrapped off and when the colour does not disappear after stretching the tissue. Due consideration must also be given to exclude other conditions that clinically appear white in colour.[8]

Figures 1-3 illustrate clinical presentations of both homogeneous and non-homogenous leukoplakias. Figure 4 illustrates a carcinoma arising in a mixed, white and red lesion.

Several tobacco-induced lesions, such as smoker's palate (Leukokeratosis nicotina palati, palatal keratosis of reverse smokers and snuff (or snus) dipper's lesion are traditionally separated from leukoplakia though they are white in appearance and are also associated with tobacco use.[5]

As leukoplakia is a provisional clinical diagnosis and the observed white patch should be subjected to a pathology review by promptly undertaking a tissue biopsy. Reasons for biopsy are to exclude other pathologies (including carcinoma) that might be responsible for the white lesion and (2) to evaluate the presence and degree of epithelial dysplasia within the lesion.[9] An additional reason is to assess any candidal colonization within the epithelium. Grade of dysplasia reported by a pathologist, in spite of controversies regarding interpretation,[10] remains our best assessment of risk for malignant transformation of OPMDs.

Proliferative verrucous leukoplakia

Any leukoplakic lesion that becomes warty, exophytic and widespread over a period of time and that has recurred after treatment should arouse clinical suspicion of proliferative verrucous leukoplakia (PVL). Widespread nature of the condition may involve multiple sites of the oral cavity primarily affecting gingiva, alveolar mucosa, tongue and buccal mucosa. Fig 5 illustrates a case of PVL with an extensive keratosis affecting the gingiva and alveolar mucosa extending to the buccal mucosa. A set of criteria for diagnosis of PVL are presented by Bagan's group.[11] The major clinical criteria they cite include: A leukoplakia lesion with more than two different oral sites, which is most frequently found in the gingiva, alveolar processes and palate. B. The existence of a verrucous area. C. That the lesions have spread or engrossed during development of the disease and D. That there has been a recurrence in a previously treated area.

All PVLs do not have a verrucoid appearance especially in the initial stages, and a group of US experts have recently ascribed a clinical diagnosis of PVL to patients presenting with multifocal lesions that are devoid of a verrucous appearance. [12] Based on the argument that multiple site involvement is a more important criterion than a verrucous appearance, Aguirre[13] has proposed an alternative term "proliferative multifocal leukoplakia" for this condition.

Erythroplakia

The term Erythroplakia is used analogously to leukoplakia and has been defined as 'a fiery red patch that cannot be characterized clinically or pathologically as any other definable disease'. The lesions of erythroplakia are usually irregular in outline though well defined and have a bright red velvety surface. Occasionally the surface is granular. The most commonly involved sub site is soft palate, as shown in Fig 6. Just as there are many oral lesions that present clinically as white patches on the mucosa, so there are a number of conditions that appear as red areas. Examples of other red patches that need to differentiated from erythroplakia are outlined by Reichart and Philipsen.[14] Two common examples often mistaken by practitioners as erythroplakia are erythematous candidiasis (denture-associated somatitis) and erythema migrans. Other conditions to include in the differential diagnosis are erosive disorders, desquamative gingivitis, discoid lupus, erosive

lichen planus, pemphigoid and other inflammatory/ infectious conditions.[14] Well-demarcated, solitary presentation of erythroplakia helps to clinically distinguish it from other more widespread disorders listed above.

A diagnostic biopsy is essential to obtain a pathologist's opinion to distinguish from above mentioned specific and nonspecific inflammatory oral lesions. This should be undertaken urgently as many erythroplakias are dysplastic or may harbour carcinoma *in situ* or even frank carcinomas. [10]

Erythroleukoplakia

Mixed white and red lesions previously referred to as speckled leukoplakia are now termed erythroleukoplakia.[1,15] The red component (Fig 2) shows either atrophy (thinning) or sometimes speckling. Erythroleukoplakia unlike leukoplakia or erythroplakia may have an irregular margin. [15] The patient may experience some soreness often due to colonization by candidal hyphae.

Oral lichen planus

The oral manifestations of lichen planus vary from subject to subject. Oral mucosal lesions are usually multiple and have a symmetrical distribution.[16] The clinical presentation of OLP can be divided into several clinical sub-types: linear, reticular, annular, papular, plaque, atrophic, and ulcerative. Bullous lichen planus is rare. In dark skinned people the affected area shows signs of pigmentation. Patients often display features of more than one subtype simultaneously.

Lichen planus lesions usually present as bilateral keratotic lace-like network of white striae on the buccal mucosa and lateral margins of tongue. Reticular type is the most frequent type encountered in clinical practice and the majority of patients are asymptomatic. The reticular lesions appear as interlaced raised lines forming a latticework (Fig 7). Sometimes the striae may have a linear or annular presentation. Reticular OLP can also be found on the mucobuccal fold, gingiva, floor of mouth, labial mucosa, lips and rarely on palate. The papular type presents as small white raised papules that the clinician must differentiate from Fordyce granules. The plaque type commonly found on the dorsum of the

tongue closely resembles leukoplakia; however, keratotic white striae are found at the lesion periphery. Atrophic erosive and ulcerative types may present as erythematous areas or with frank ulceration. Often keratotic white striae are seen at the margins. When ulcerated, patients typically complain of soreness or a burning sensation while eating hot or spicy food.

Atrophic OLP presenting on the gingivae, can be seen as desquamative gingivitis. The bullous type is rare, tend to recur and its important to differentiate this type from pemphigus or mucous membrane pemphigoid.

Some patients may exhibit cutaneous lichen planus and the medical history may help in identification of OLP cases. Other extra-oral mucosal sites such as genitalia may also be affected. Genital examination may help to identify persons with vulvo-vaginal gingival (VVG) variant of lichen planus.

Lichen planus is usually diagnosed clinically. Its bilateral distribution and the presence of the classic reticular forms with keratotic white striae are helpful for a chair-side diagnosis. [17] The differential diagnosis for OLP when it presents with a reticular/ erythematous appearance includes lichenoid lesions, lichen sclerosus, lupus eruthematosus (DLE), chronic ulcerative stomatitis and when plaque like includes oral leukoplakia. Biopsy and histopathological examination are recommended to make a definitive diagnosis. The essential histologic findings are comparable, regardless of the areas involved or the sub type of clinical presentation. Microscopy also aids to identify presence of epithelial dysplasia, and on rare occasions malignancy. Direct immunofluorescence studies do not aid the diagnosis of lichen planus, but could assist to rule out DLE or mucous membrane pemphigoid.

OLP may last for several years, with periods of symptoms and remission. In patients with the ulcerative type of OLP sclerotic fibrous bands may appear. A systematic review has confirmed malignant transformation in OLP lesions [18] but there are no specific criteria to assess this risk.

Oral Lichenoid Lesions

Oral lichenoid lesions (OLL) are intraoral white and red lesions with reticular striated appearances sharing similar clinical features as oral lichen planus (OLP), but having an underlying causative agent. Another term used analogously is oral lichenoid reactions (OLR). OLL/OLR can be classified into three types: 1) in topographic relationship to a dental restoration [19], often amalgam, also named as oral lichenoid contact lesions (OLCR) 2) drug-related, 3) in association to chronic graft versus host disease (cGVHD).

OLL/OLR presents as white or mixed white and red lesions, sometimes with additional ulceration. Associated clinical signs are white reticular, linear or annular striae and/or white plaque-like patches. Red and mixed lesions appear as erythematous atrophic patches often with some ulceration of the oral mucosa, known as erosive type of OLL/OLR. The differentiation from OLP may be clinically difficult in some cases.

OLL/OLR due to hypersensitivity to dental restorations is however, often localized to the site in contact with the allergenic material (Fig 8), while OLP has a bilateral and widespread presentation. OLL/OLR induced due to a reaction to drugs show various clinically features, with a certain tendency of being unilateral and erosive. OLRs also occur in betel quid chewers at the site of placement of betel quid as illustrated in Fig 9

The diagnosis of OLP/ OLL/OLR is mostly based on the synthesis between history, clinical examination, a patch test when indicated [20] and by microscopy. For OLL/OLR suspicious to be drug-induced the history of any correlation between the beginning of the intake of the medication and first symptoms may also be informative, even though reactions can occur several weeks or months after the prescription. However, the distinction between OLL/OLR and OLP is often difficult under the microscope and there is no universal agreement among pathologists to distinguish these two entities. Possible malignant transformation in OLL was first described in a case series from the Netherlands. [21]

Graft versus Host Disease (cGVHD)

GVHD is a complication arising in recipients of allogenic hematopoietic stem cells or bone marrow transplantation. Chronis GVHD is a systemic condition with a wide variety of signs and symptoms and affects many organ sites. The oral cavity is one of the most frequently affected sites.

GVHD can be widespread in the mouth. The primary symptom relates to soreness at mealtimes. The disease presents with keratotic striations, white plaques or erosive and ulcerative areas of the oral cavity.[22] There is typically an involvement of the buccal mucosa and lateral tongue. The dorsum of the tongue may show papillary atrophy. Other clinical features include xerostomia (oral dryness) and patients may develop recurrent mucoceles on labial and buccal mucosae, tongue or soft palate. Malignant transformation is reported in follow up studies.[23]

Discoid lupus erythematosus (DLE)

Lupus erythematosus is a chronic autoimmune disease which can be subdivided into three forms; the systemic, drug-induced and discoid. It is the latter benign variant that commonly affects the skin and may involve the mucosal surface of lips and the oral cavity. Around 20% patients with systemic lupus may also manifest with oral lesions. The disease is driven by an immune complex deposition in affected sites, leading to vasculitis.

The discoid variety typically affect s the solar exposed areas of face and neck and may present with the typical butterfly rash across the nasal bridge. The oral lesions consist of central zones of ulceration or erythema (representing vasculitis) surrounded by white striations, bearing a close resemblance to oral lichen planus (Fig 10). Immunofluorescence studies demonstrate subepithelial immunoglobulin and complement deposition (the lupus band) [24] which assist in distinguishing DLE from Lichen planus. During resolution erosive areas of DLE may lead to postinflammatoy pigmentation. Oral lesions typically affect the buccal mucosa, palate and lips. The lower lip is the most commonly affected site in DLE-related malignant transformation. DLE has been recognised by the Collaborating Centre of the World Health Organization as a potentially malignant disorder[1] but malignant transformation is known to be exceedingly rare. In a recent review of the literature Arvanitidou et al [25] documented 21 reported cases of carcinomas of the vermillion border of

the lip arising in DLE lesions, and added a further case of their own. DLE-related SCCs have been observed to be more aggressive than conventional lip cancers. [26]

Oral Submucous Fibrosis

Oral submucous fibrosis (OSF) is a chronic, insidious disease that affects the lamina propria of the oral mucosa and as the disease advances it involves tissues deeper in the submucosa of the oral cavity with resulting loss of fibroelasticity. History of betel quid and areca nut chewing in an Asian patient who has limited opening the mouth should arouse suspicion of this condition.

The disease is characterized by the presence leathery mucosal texture and palpable vertical fibrous bands in the oral mucosa, ultimately leading to limitation of mouth opening and rigidity of the tongue.[27] Early features include blanching of mucosa (Fig 11) , loss of normal pigmentation and a burning sensation of the mucosa to spicy food.[28] On clinical examination, sunken cheeks and limitation of opening the mouth may be obvious. In addition, the tongue may be small, exhibit limited mobility and show a marked loss of papillae. The palate may appear pale with horizontal bands across the soft palate (Fig 12) and uvula may be shrunken or deformed. The severity and permutations of signs and symptoms of OSF are highly variable. The severity of the disease is generally measured objectively by assessing the mouth opening. [29] and by the presence of leukoplakia or erythroplakia as multiple lesions. Progressive limitation of mouth opening is a hallmark feature of this disease and OSF has a significant impact on the quality of life of affected individuals.[30]

Among betel quid users, particularly in association with OSF a new lesion with malignant potential has been described as “oral verrucous hyperplasia.[31] In a consensus report by a panel of South Asian pathologists [29] this “mass type” novel lesion observed in South Asian betel quid chewers which has both exophytic and verrucous phenotypes, has been termed - Exophytic oral verrucous hyperplasia. Further studies have complemented their findings.[33]

Palatal changes of reverse smokers

In this unusual form of smoking, referred to as reverse smoking, the lighted end of a cigar, chutta (an Indian smoking product) or cigarette is placed inside the mouth. The habit is prevalent in parts of India, the Caribbean Islands, Colombia, Panama, Venezuela, Jamaica, Sardinia and the Philippines. The observed mucosal changes associated with reverse smoking were comprehensively described in a 10-year follow-up study of Indian villagers by Gupta et al. [34] The mucosal changes that were described in reverse chutta smokers included, thickened leukoplakic plaques of palate, mucosal nodularity, excrescences around orifices of palatal (minor)mucosal glands, yellowish brown staining, erythema and ulceration. The changes noted involved most of the palatal surface exposed to direct heat and smoke. Comparable palatal lesions were noted among Filipino women [35] and from a territory in Colombia among persons with similar habits. [36] Follow-up studies reported from India[34] clearly demonstrated the potentially malignant nature of this condition as 6 persons in a cohort of close to 3000 subjects over 6 years developed palatal cancer. Reverse smokers' palatal lesions are more persistent than leukokeratosis nicotina palati lesions found in regular cigarette smokers (referred to earlier) and compared with leukoplakia have a higher hazard ratio to develop malignancies.

Epidermolysis bullosa

Epidermolysis bullosa (EB) is a skin disease characterized by epithelial fragility that may manifest with blistering and erosions of the oral mucosa. The disease is classified into 32 different subtypes. Intraoral soft tissue manifestations are found in all subtypes and include marked frequency of oral and perioral blistering leading to ulceration, scaring and obliteration of the oral vestibule and microstomia. [37]

Fine et al [38] conducted an analysis of a data set on 2745 EB patients entered on the National EB Registry in the continental United States (1986-2002). At least one SCC arose in 2.6% (73/2745) of the study population almost all in sun-exposed areas. Multiple SCCs were found in the group with recessive dystrophic EB (RDEB). Based on this analysis the authors highlighted that in the recessive dystrophic type (RDEB) the life time risk of developing

squamous epidermal cancers is greater than 90% . Only one oral SCC was reported on the tongue among non-cutaneous cancers. [38]

Others have published case reports or small case series of oral squamous cell carcinomas particularly among individuals with severe generalized RDEB, reviewed by Wright. [39] Malignancies in EB patients can occur in the third decade of life or even earlier. Due to increased risk of cancer EB is included as a potentially malignant disorder, though specific oral premalignant lesions associated with EB are not well characterised in the literature. As patients with EB thus may be at an increased risk of oral squamous cell carcinoma, during oral examination it is prudent to be extra vigilant in monitoring changes for any suspicious features around any oral ulcers which are so frequent in this condition.

Dyskeratosis congenita

Dyskeratosis congenita (DC) is a rare inherited bone marrow failure syndrome and patients with DC have significantly increased risk of malignancy. Oral leukoplakia is the most common presentation in this condition, found in 65% to 80% of patients.[40] Leukoplakic patches, of the dorsal tongue and sometimes on the buccal mucosa[41] are features of the classic triad of signs that include lacy reticular hyperpigmentation of the skin and nail dystrophy. The tongue is affected often from a young age and most reported cases with oral leukoplakia have been in children and adolescents under the age of 15 years.[42] One of the early descriptions of oral leukoplakia in DC was in a 10 year old boy reported by Ogden in this journal.[43] Oral white lesions are rare in children and the identification of a white patch on the tongue of a child, in the absence of any other obvious cause eg. candidal infection or chronic trauma must arouse suspicion of this rare condition. The condition is attributed to several mutations of genes that help to maintain telomeres such as the DKC1 gene, which encodes for the protein dyskerin.[44] These leukoplakic patches of the mouth in DC patients have a significantly increased risk of developing to squamous cell cancers.

Actinic cheilitis

Actinic cheilitis (AC) is a chronic inflammatory condition of the lip that results from excessive exposure to solar ultraviolet (UV) radiation and most often affects the lower lip. Those with fairer skin are at a heightened risk,[45] may predispose to AC and men show a stronger predisposition for AC than females.

AC has a wide range of clinical features. Common clinical presentations comprise of white lesions in conjunction with crusting, flaking, dryness or of a mottled appearance indicating the simultaneous presence of erythema and white patches.[46] During the course of the disease ulcerative lesions may develop with inflammation, atrophy and loss of epithelium. Sun exposure is the most important risk factor for AC. The development of AC is dose-dependent and is associated with patient's solar exposure, age, genetic predisposition, geographic latitude of residence, out-door occupation, leisure activities, and non-use of lip protective agents.[47]

Squamous cell carcinoma of Lip is often found in a background of actinic cheilitis. However, evolution of SCC from AC has not been studied in detail by careful follow up, except in one Greek study. Markopoulos et al[48] reported 65 patients with AC and on close follow up 11 persons (16.9%) developed lip cancer. Progress of AC can be minimized by the use of an appropriate sun screen when outdoors.

Conclusion:

OPMDs have an increased risk of developing into oral cancer. Several varieties are recognised. Some of them are solitary lesions while others referred to as conditions are multifocal or widespread within the oral cavity. Leukoplakia is the most common OPMD encountered in clinical practice. For patients with a clinically evident oral mucosal lesion considered to be suspicious of an OPMD, clinicians should perform a biopsy of the lesion or provide immediate referral to a specialist.

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Legends to Figures

Fig 1. A patch of homogeneous leukoplakia with a flat, thin and a uniformly white appearance affecting ventrolateral tongue. The colour and the appearance mimicks white paint brushed onto the mucosa.

Fig 2. A patch of erythroleukoplakia affecting lateral margin of tongue with mixed white and red areas within the lesion. Note, the boarders are irregular.

Fig 3. A small patch of verrucous leukoplakia over the attached gingiva. Note the corrugated, verrucous appearance of the lesion.

Fig 4. A carcinoma arising in a patch of non-homogeneous leukoplakia.

Fig 5. Proliferative verrucous leukoplakia affecting gingiva, alveolar and buccal mucosa.

Fig 6. An erythroplakia affecting the soft palate. On biopsy severe dysplasia was reported in this red patch.

Fig 7. Reticular oral lichen planus involving posterior part of the oral cavity.

Fig 8. Oral lichenoid lesions in close proximity to amalgam restorations on lower molar teeth.

Fig 9. Oral lichenoid reaction to betel quid in an Asian gentleman who regularly parked the quid in this location. Note staining on teeth from areca nut.

Fig 10. A lupus patch on the palate of a patient diagnosed with systemic lupus erythematosus. The affected area shows irregular keratosis surrounding a zone of central eryhtma, with some radiating white striae.

Fig 11. Blanching of buccal mucosa in a case of early oral submucous fibrosis

Fig 12. Palatal fibrosis seen in a patient with moderately advanced oral submucous fibrosis

Fig 1.jpg

Fig 10.jpg

Fig 11.jpg

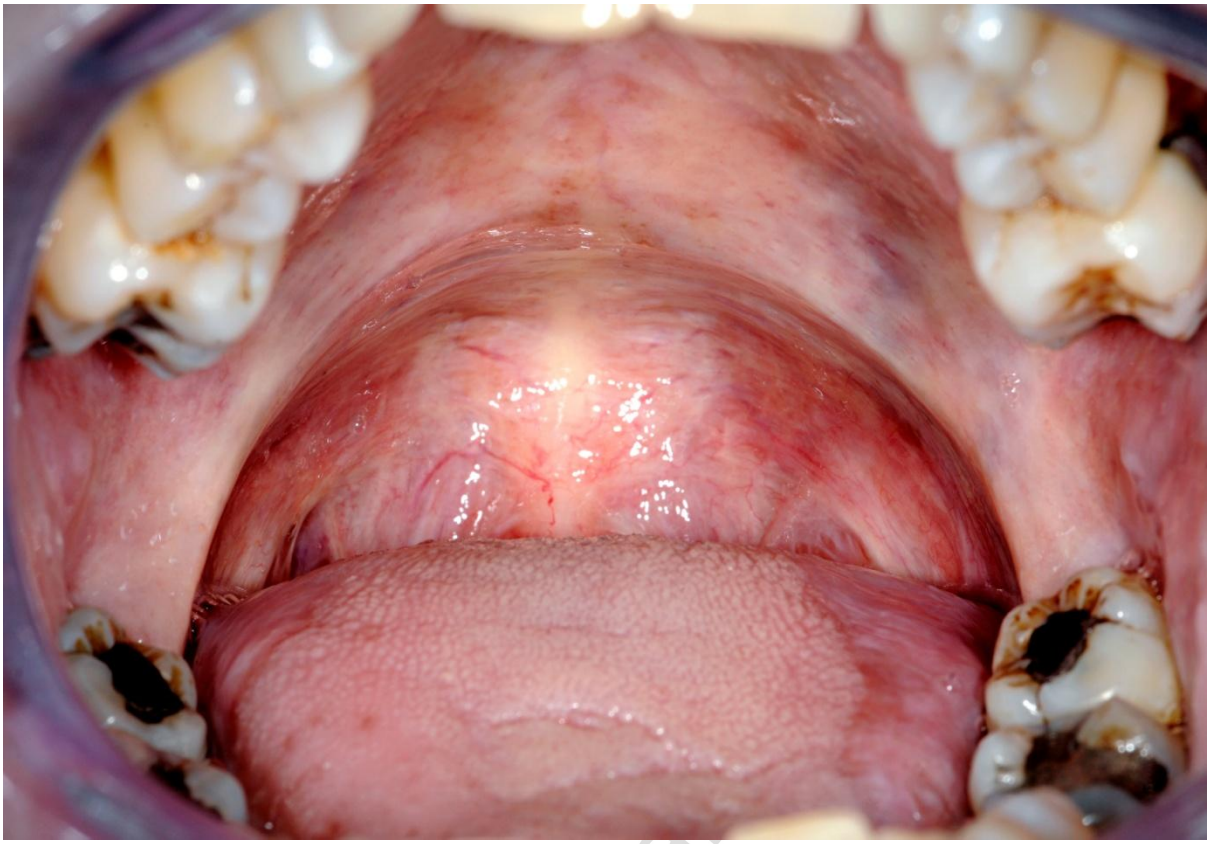

Fig 12.jpg

Fig 2.jpg

Fig 3.jpg

Fig 4.jpg

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Fig 6.jpg

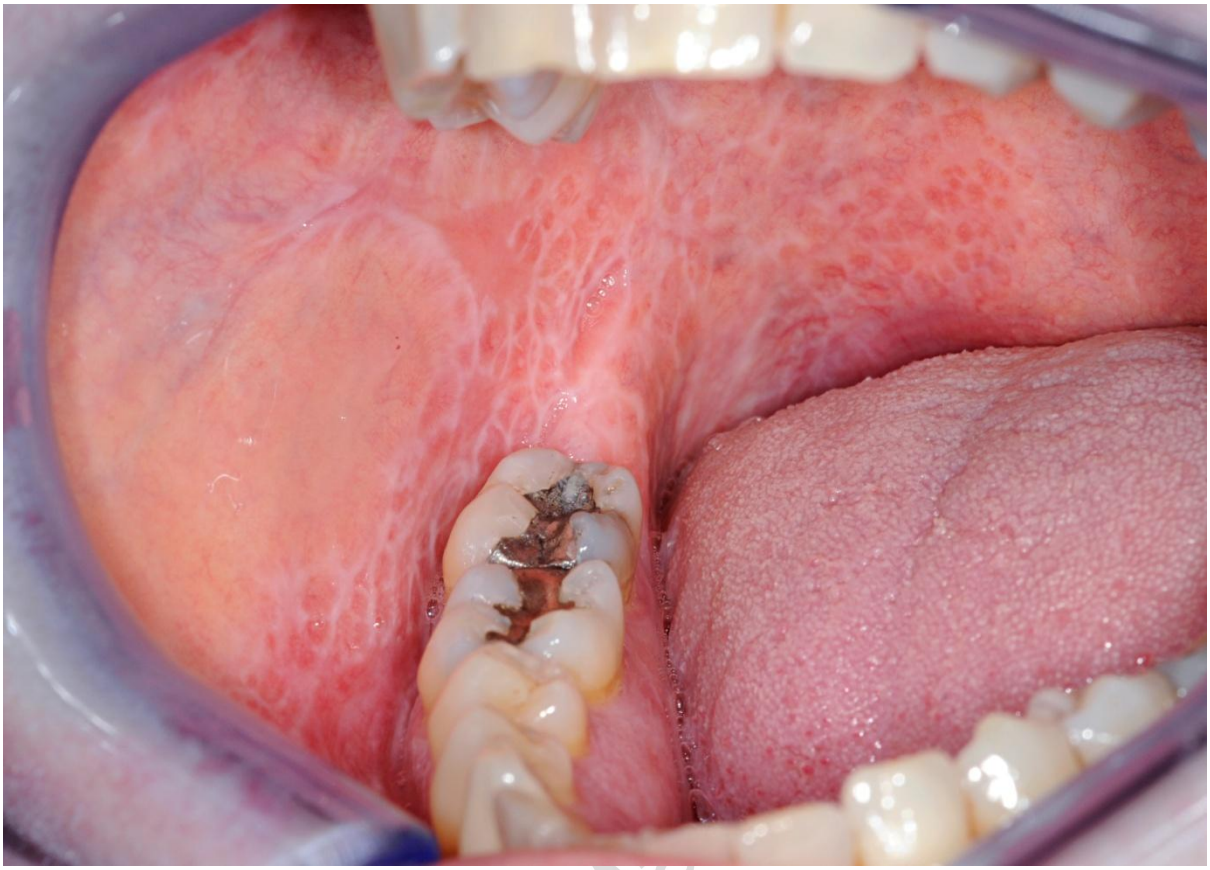

Fig 7.jpg

Fig 8.jpg

Fig 9.jpg